

Fig. 1

A) Example of acute phase response protein

B) Example of coagulation-fibrinolytic protein

C) Example of disinfectant protein

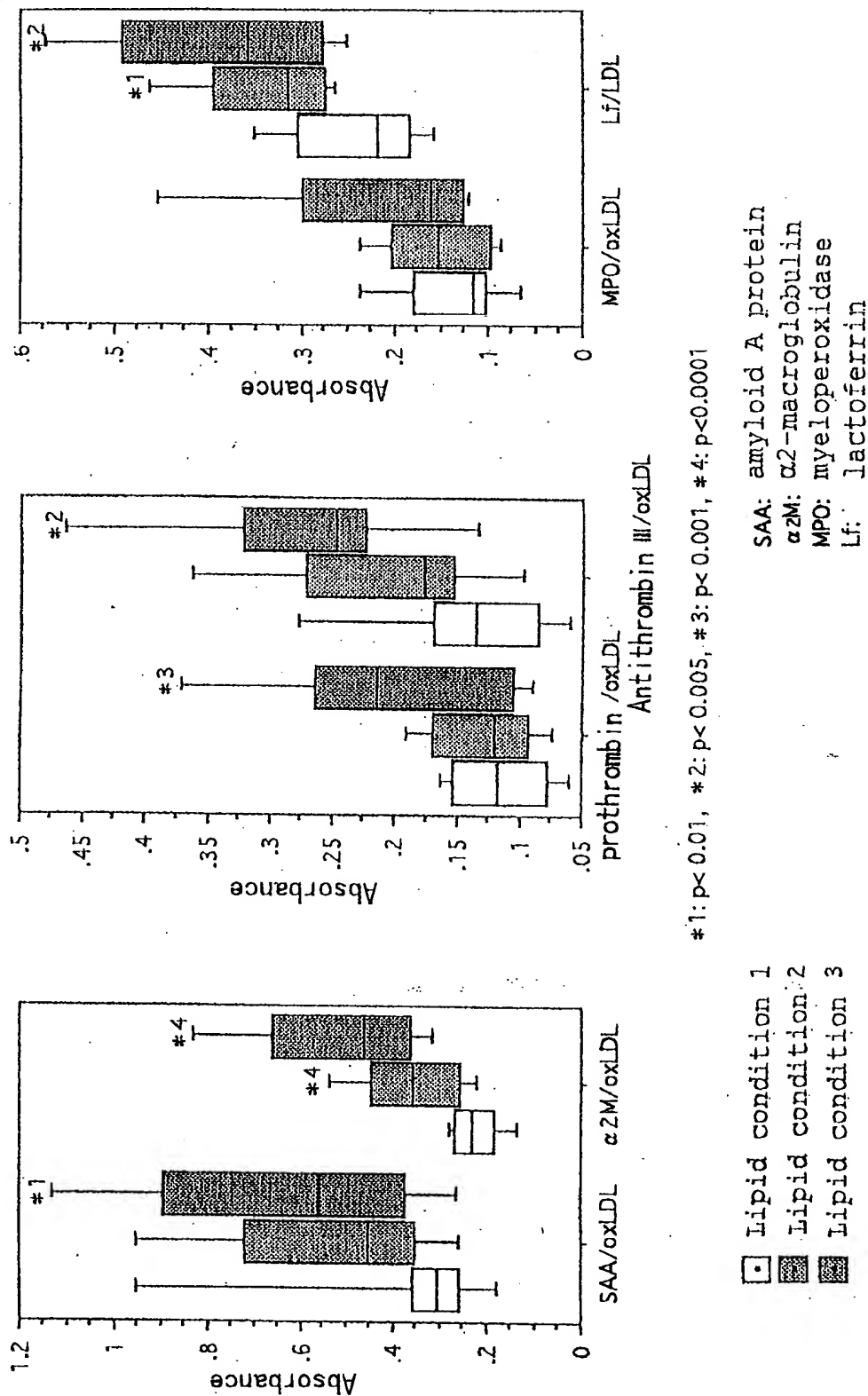
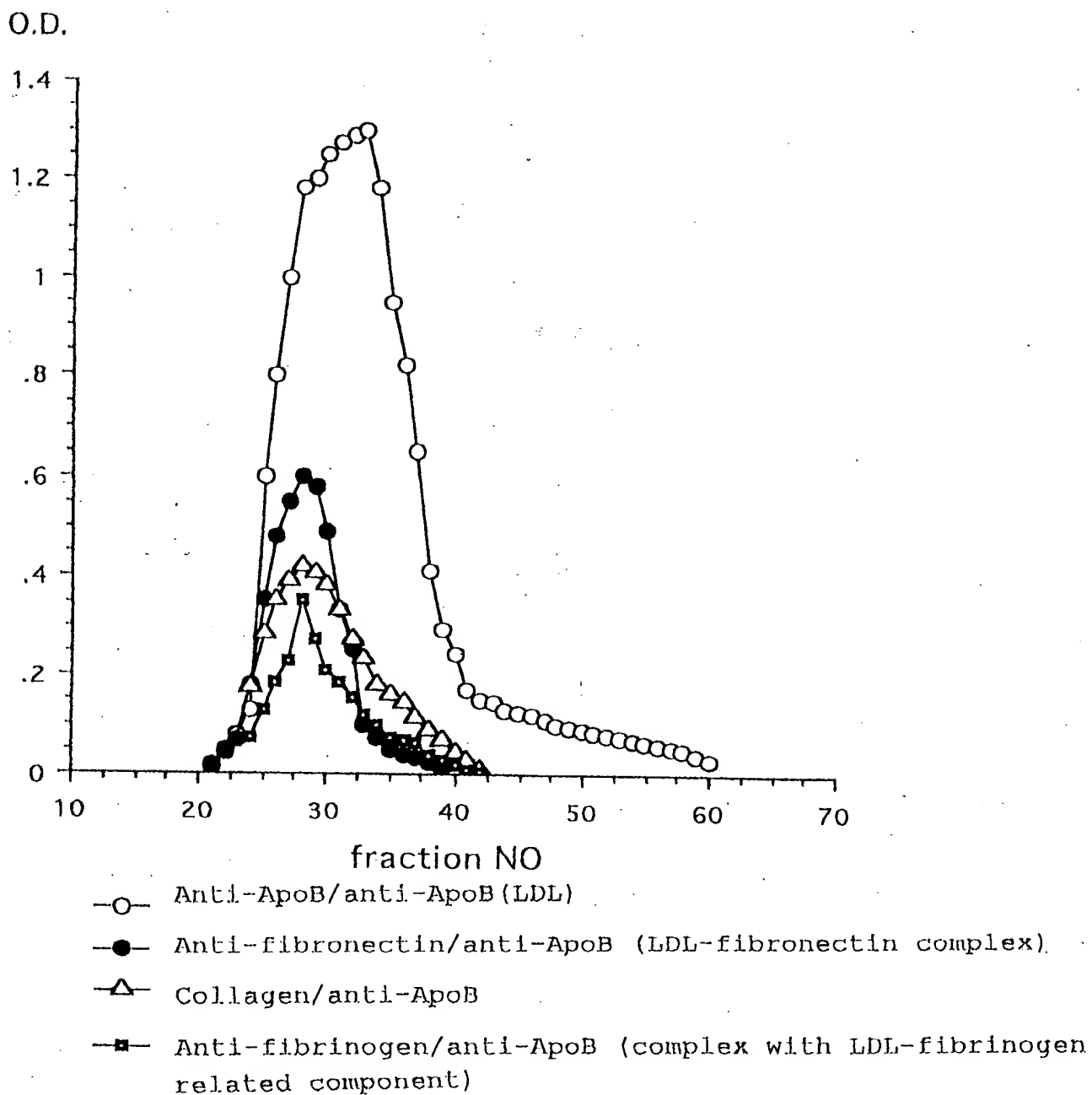


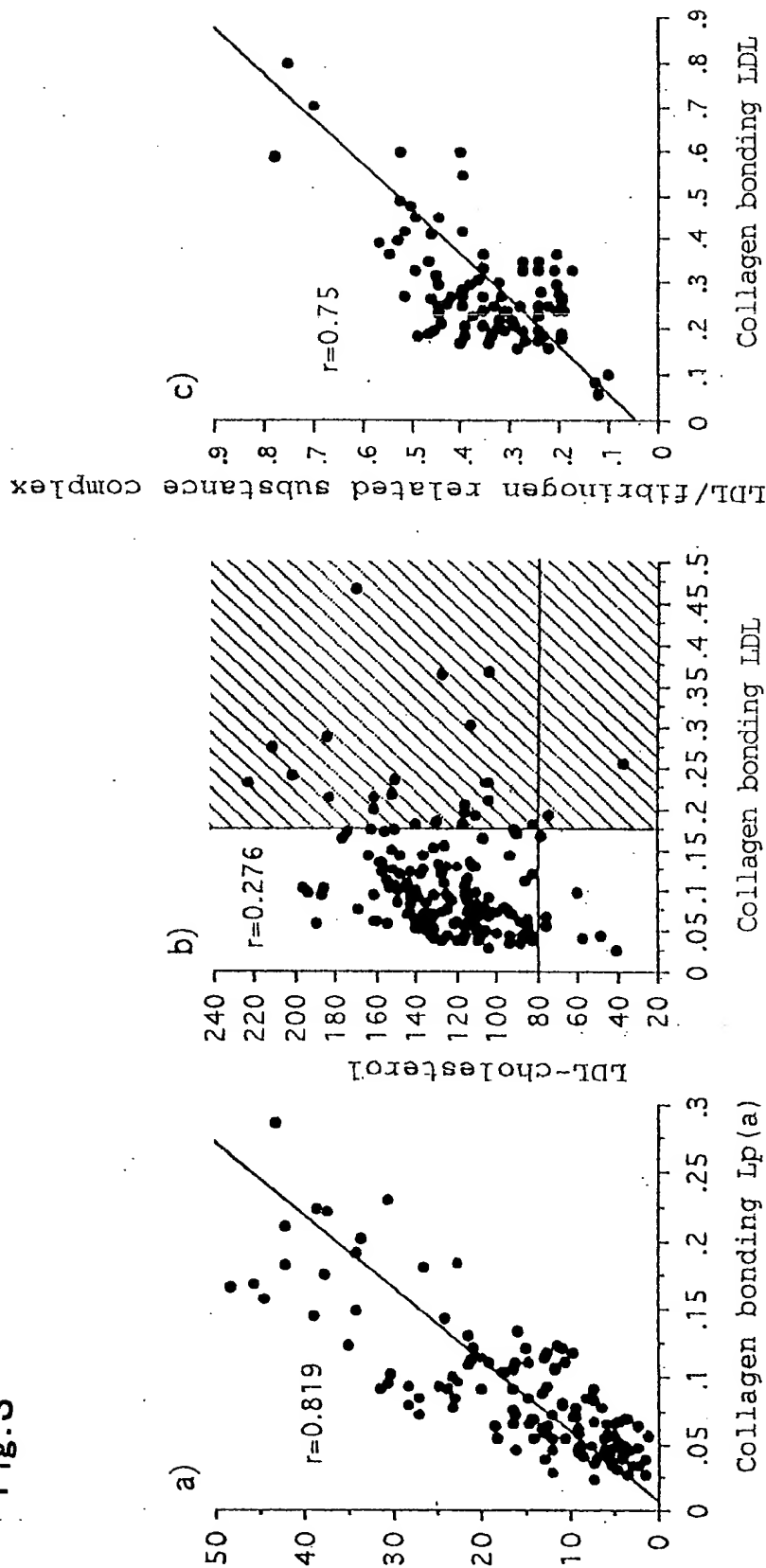
Fig. 1 Comparison of concentrations of complexes of LDL or denatured LDL with acute phase response protein (A), coagulation-fibrinolytic system protein (B) and disinfectant protein (C), among three groups different in blood lipid concentration

Fig.2



LDL-fibrinogen related component, LDL-fibronectin complex and collagen bonding lipoprotein, present in human serum LDL fraction

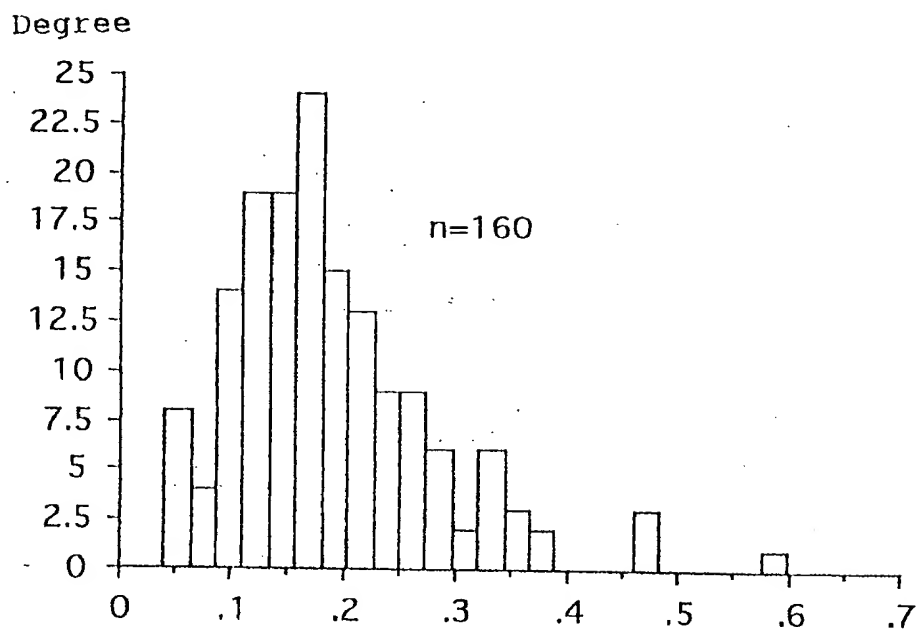
Fig.3



According to a novel arteriosclerosis onset mechanism hypothesis (hypothesis of mechanism initiating from lipid precipitation under blood endothelium) according to Williams et al. (Arterioscler, Trom., 15: 551, 1995), it is indicated that when the concentration of blood LDL cholesterol is 80mg/d or more, lipid precipitation under blood endothelium occurs, while the present inventors have found that the presence of novel lipoprotein (extracellular substrate component bonding lipoprotein: collagen bonding LDL and the like, including a complex with a LDL-fibrinogen related substance) concerning arteriosclerosis disease likewise as Lp(a) in blood is essential for the occurrence of lipid precipitation under blood endothelium. (In Fig. 2b, cases presented in diagonal portions are examples of positive results of novel lipoprotein concerning arteriosclerosis disease)

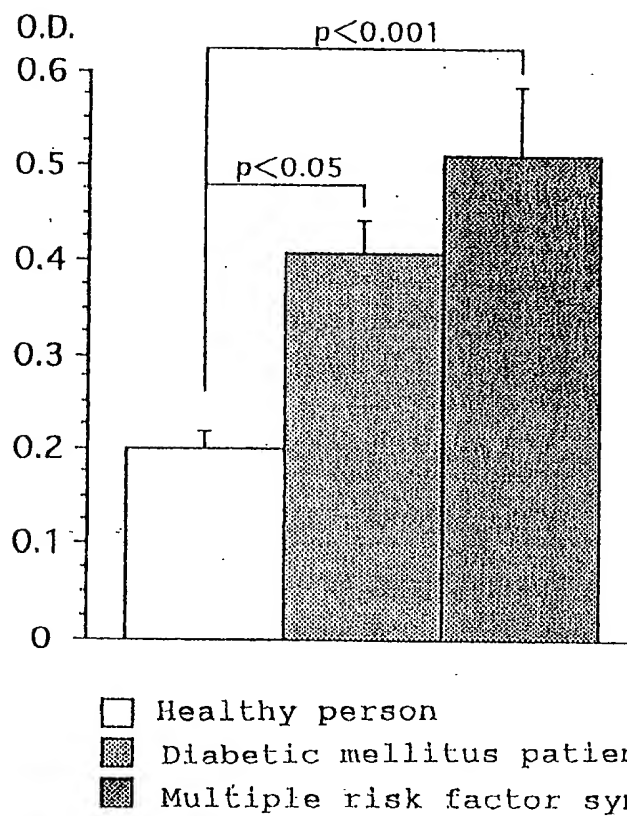
Relationship between blood Lp(a) concentration and extracellular substrate-protein (collagen) bonding Lp(a) concentration, relationship between blood LDL-cholesterol concentration and concentration of novel lipoprotein concerning arteriosclerotic lesion, and relationship between concentration of complex with LDL-fibrinogen related substance and concentration of collagen bonding LDL

Fig.4



Distribution of concentration of LDL-fibrinogen related substance complex in serum of healthy person

Fig.5



Comparison of amounts of LDL-fibrinogen related substance complex in healthy person, diabetic mellitus patient and multiple risk factor syndrome

Fig.6

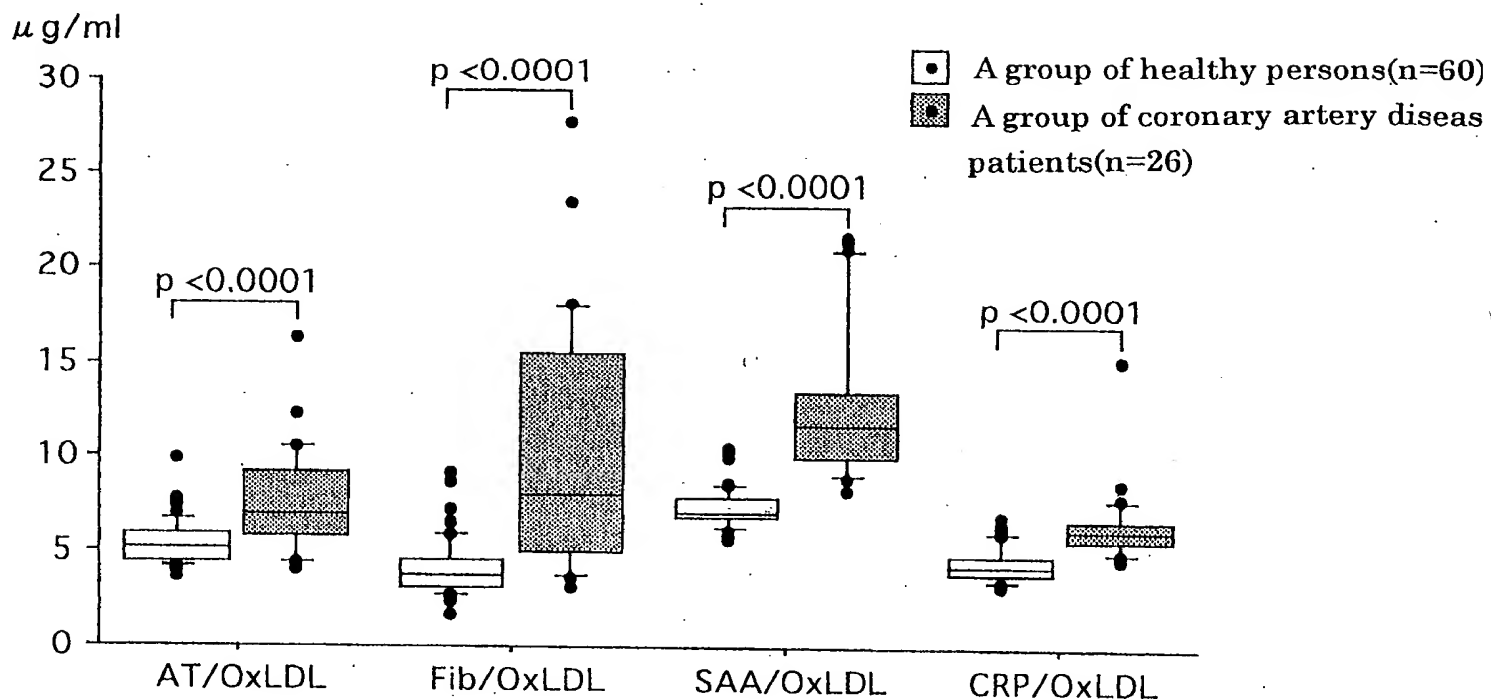


Fig. 6 Distribution of concentrations of AT/OxLDL, fib/OxLDL, SAA/OxLDL, CRP/OxLDL complexes in the serums of a group of healthy persons(those taking health examinations) and a group of coronary artery disease patients(those found by photograph examination with more than 50% stricture in their main coronary arteries)

Fig.7

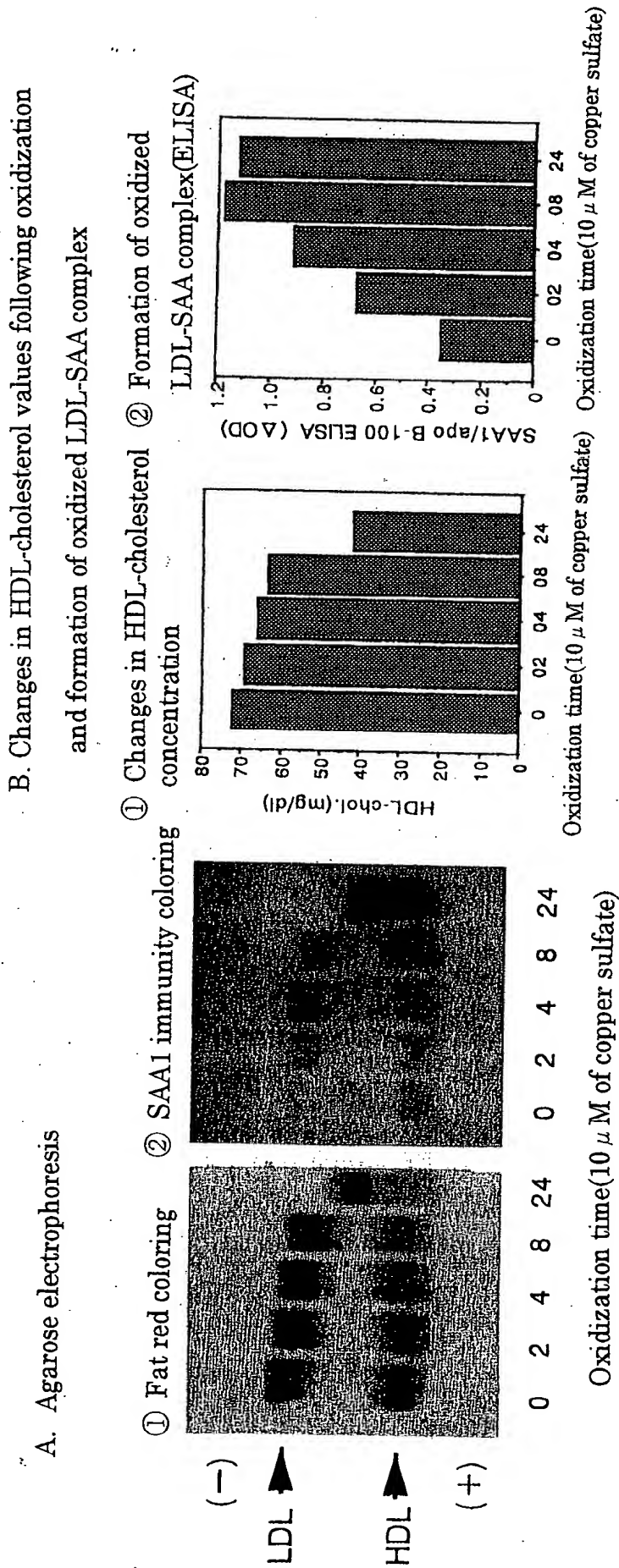


Fig. 7 Review of the formation mechanism of oxidized LDL-(serum amyloid A1;SAA1) complex

After equal amounts of native LDL and native HDL were mixed, 10 μ M of copper sulfate was added, and the mixture was left at 37°C. Oxidized LDL-SAA complex was formed in accordance with the degree of oxidation(Fig. 7, A-②, B-②). On the other hand, HDL-cholesterol values lowered following oxidation(Fig. 7, B①).